

Invasion lags: The stories we tell ourselves and our inability to infer process from pattern

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Abstract

Aim: Many alien species experience a lag phase between arriving in a region and becoming invasive, which can provide a valuable window of opportunity for management. Our ability to predict which species are experiencing lags has major implications for management decisions that are worth billions of dollars and that may determine the survival of some native species. To date, timing and causes of lag and release have been identified post hoc, based on historical narratives.

Location: Global.

Methods: We use a simple but realistic simulation of population spread over a fragmented landscape. To break the invasion lag, we introduce a sudden, discrete change in dispersal.

Results: We show that the ability to predict invasion lags is minimal even under controlled circumstances. We also show a non-negligible risk of falsely attributing lag breaks to mechanisms based on invasion trajectories and coincidences in timing.

Main conclusions: We suggest that post hoc narratives may lead us to erroneously believe we can predict lags and that a precautionary approach is the only sound management practice for most alien species.

KEYWORDS

alien species, dispersal, fragmentation, invasive species, lag phase, population spread, sleeper weeds

1 | INTRODUCTION

Most alien species that arrive in a new region either fail to establish or persist as minor components of ecological communities without ever becoming invasive (Williamson & Fitter, 1996). However, those species that do become invasive can cause enormous economic damage and threaten native biodiversity (Pimentel, Zuniga, & Morrison, 2005). The cheapest, and often only effective time to control an invasive population is during the lag phase, the time between a species arriving in a region and its population expanding and being considered invasive (Crooks, 2005).

There are many documented examples of lag phases among invasive species, and when lag phases do occur they can stretch over long time periods. Among 142 invasive plant species in New Zealand,

91% had a statistically discernible lag phase, and 5% of these species were present for more than 40 years before becoming invasive (Aikio, Duncan, & Hulme, 2010). Similarly, a lag phase was found in the majority of 120 exotic plant species in the midwestern USA, with lag times ranging from 3 to 140 years (Larkin, 2012), and in 14 of 17 invasive bird species in the Hawaiian archipelago, with lag times ranging from 10 to 38 years (Aagaard & Lockwood, 2014). The mean lag between first record and naturalization in 77 species of tropical grasses in northern Australia was 27.4 years, with a maximum of 124 years (van Klinken, Panetta, Coutts, & Simon, 2015).

Lag phases provide windows of opportunity for management (Simberloff, 2003). Devoting resources to early detection and eradication is the most cost-effective management strategy under a wide range of conditions (Epanchin-Niell, 2017; Holden, Nyrop, & Ellner,

2016). However, it is only optimal to control a species before it becomes widespread if it would go on to become widespread and damaging without management (Eparchin-Niell, 2017; Hauser & McCarthy, 2009). Unpredictable invasion lags will make it harder to discriminate between those species that will never be problematic and those that have been benign to date, but will become invasive, so-called “sleepers weeds” (Groves, 2006). The consequences of getting this prediction wrong, and ignoring a species that become invasive, can be profound (Simberloff, 2003).

Predicting invasive potential requires understanding which environmental or ecological changes are likely to break lag phases. This is difficult because the lag phase for any one species is likely to be caused by multiple interacting factors (Crooks, 2005). The most direct way to disentangle the effect of these interacting factors is a controlled experiment. However, the temporal and spatial scale required for any field experiment examining invasion lags, along with ethical concerns over releasing potentially invasive species, mean that manipulative field experiments on invasion lags have not been carried out.

Because manipulative field experiments are infeasible, the main tool used to understand invasion lags has been examining historical examples. Historical invasion lags have been attributed to habitat or climate change (Kowarik, 1995), slow dispersal of highly competitive species (Bennett, Vellend, Lilley, Cornwell, & Arcese, 2013), Allee effects (Taylor & Hastings, 2005), introduction of pollinators (Amodeo & Zalba, 2013; Nadel, Frank, & Knight, 1992), evolution of novel traits (Perkins, Phillips, Baskett, & Hastings, 2013) and hybridization (Mukherjee et al., 2012). In some cases, post hoc explanations for observed invasion lags can invoke mechanisms that would have been extremely difficult to predict *a priori*. For example, the expansion of disturbance-tolerant species in Europe has been attributed to World War II (Baker, 1974; Kowarik, 1995). Many of these narratives can offer plausible explanations for the observed lag and release. However, given that they are post hoc reconstructions rather than testable hypotheses, it is unclear whether they can accurately determine which mechanisms actually caused the lag phase and its break, and therefore, how they can inform a general predictive framework.

In ecology, simulation models have been used to identify potentially important processes in systems that operate at temporal and spatial scales which make direct study difficult (Coutts, van Klinken, Yokomizo, & Buckley, 2011; Zurell et al., 2010). Simulation models also have an important role in testing what can be reasonably inferred from data. If we use the simulation to generate the data, then we know the “truth,” and can test our observations of that data against this “truth” (Zurell et al., 2010).

Our aim was to test how variable invasion lags might be in a simplified, simulated system, and whether the mechanism behind a break-in lag phase can be determined through a coincidence in timing. Invasion lags can be caused by multiple mechanisms; we focus on two commonly cited ones: an increase in dispersal ability (Crooks, 2005) and fragmented habitat (Bennett et al., 2013). Dispersal ability (particularly in long-distance dispersal ability) has been found to greatly influence the speed of historical invasions (Clark, Macklin, & Wood, 1998). Fragmented habitat inhibits spread until a propagule establishes a population in a large patch of suitable habitat. Thus, a lag break may

occur from stochastic spread with no change in landscape or dispersal ability. We set up a simulated system and then examine how changes in dispersal ability impact population spread on average and across individual iterations. In effect, we simulate a simplified, controlled, replicated, invasion lag experiment that we cannot carry out in reality.

2 | METHODS

We test how fragmentation-induced lags impact population spread and determine how variable these lag times can be. We then test how effectively the variability in fragmentation-induced lag phases masks a break-in invasion lag caused by the change in the dispersal kernel. We use a spatially explicit population model for a generic woody plant, where the population spreads over a fragmented landscape. See Appendix S1 and Coutts et al. (2011) for further explanation of the model.

Each simulation begins in the same way, with a single individual in either the centre-most southern cell that is suitable or a random suitable cell. We then simulate the population spread under two phases. The population spreads for 50 time steps under an initial dispersal kernel and then the dispersal pattern changes (see Figure 1). The shift in dispersal represents some change which may increase dispersal ability, such as the introduction of a new dispersal vector (Crooks, 2005) or the rapid evolution of dispersal traits (Perkins et al., 2013). The model represents a simple case where two interacting processes (habitat fragmentation and

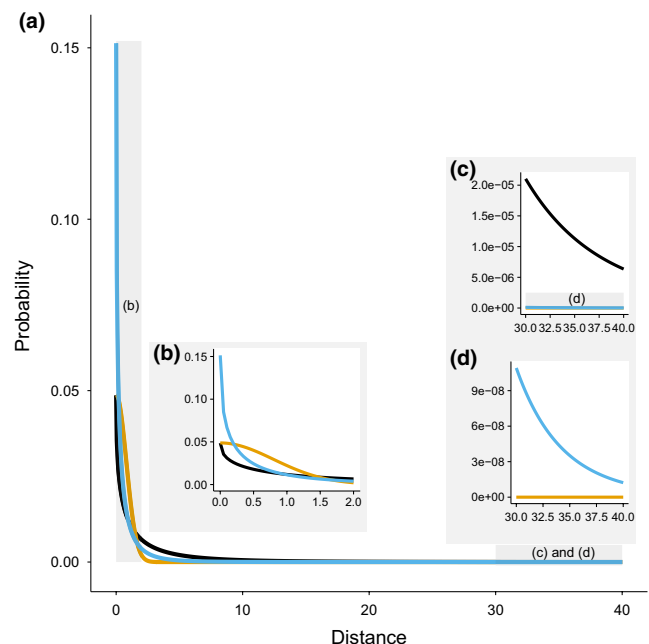


FIGURE 1 2Dt dispersal kernels for restricted dispersal ($\mu = 1$, $c = 2$; amber), less restricted dispersal ($\mu = 3$, $c = 0.5$; blue) and high dispersal ($\mu = 10$, $c = 2$; black). Insets reveal more detail at distances < 2 m (b) and in the tail of the kernel (c, d). Regions expanded in the insets are marked grey on a). The difference between the tails of the high dispersal kernel (black) and the two restricted kernels (blue, amber) is highlighted in c). The difference between the tails of the restricted (amber) and less restricted (blue) kernels is highlighted in d). [Colour figure can be viewed at wileyonlinelibrary.com]

TABLE 1 Parameters used in the sensitivity analysis, and the levels at which they were tested. Levels are the minimum, mid-range and maximum values seen in the literature for invasive woody species used to parameterize the model in Coutts et al. (2011; appendix 2). The exception is m , age of maturity, which only used studies of shorter-lived woody species. Very long times to maturity would induce an invasion lag at the start of the invasion due to all individuals being the same age. In this study, we want only fragmentation and dispersal limitation to drive invasion lags

Parameter	Value	Levels	Interpretation
f	32	32, 132	Number of offspring that germinate and survive, per adult per time step
s	0.7, 0.95	0.7, 0.99	Probability that an established individual survives one time step
m	3	1, 5	Age that individual's first start producing offspring
r	Variable	0.25, 0.5	Proportion of cells that are suitable habitat

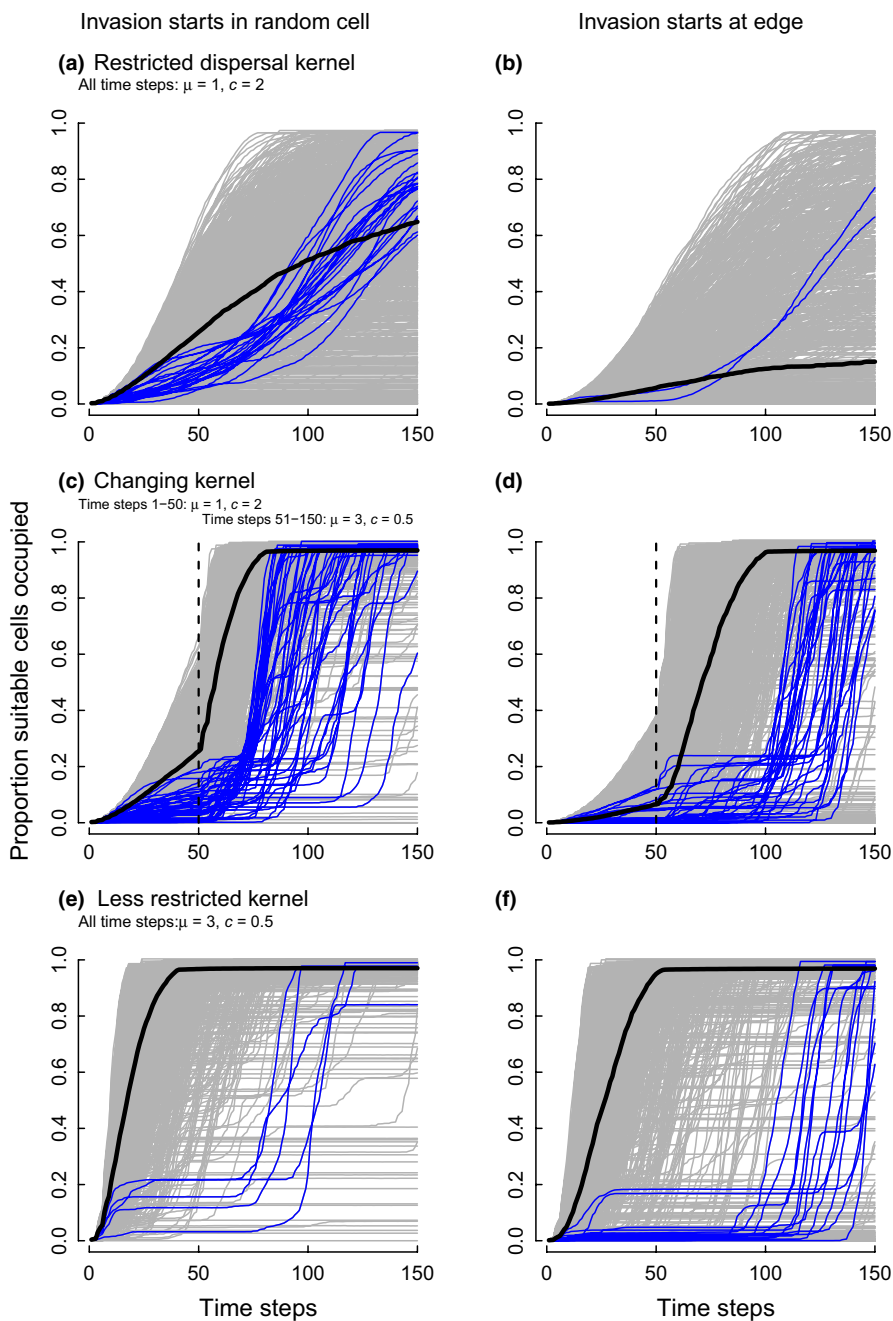


FIGURE 2 Population trajectories for 1,000 simulations (grey lines) under three dispersal scenarios: (a, b) restricted only; (c, d) change from restricted to less restricted; and (e, f) less restricted only. μ is the mean dispersal distance in cells, and c is a shape parameter that controls the thickness of the kernel tail. The invasion either starts in a random cell (a, c, d) or at the edge (b, d, f). Age of first seed production is 3, survival probability is .95, proportion of suitable habitat is 0.25, and new established plants per adult per time step (effective fecundity) is 32. The dotted line shows when the kernel changed after 50 time steps. Black line shows the median of all 1,000 simulations. Blue lines highlight long invasion lags, where <25% of suitable habitat was occupied at 70 time steps, and >60% of suitable habitat was occupied at 150 time steps. [Colour figure can be viewed at wileyonlinelibrary.com]

change in dispersal kernel) can cause invasion lags. As such, it represents a best-case scenario for being able to link the timing of an invasion lag to a mechanism (dispersal limitation release).

This simplified population model allows us to test our questions under ideal circumstances, with as few population processes as possible to influence the invasion lag. The model has no seed bank, and the number of

seeds produced, the probability that a seed germinates, establishes and survives at least 1 year is combined into a single effective fecundity parameter (f). Once an individual has survived 1 year, juveniles and adults survive with the same probability (s). Individuals start to produce seeds at age (m). See Table 1 for a list of parameter values.

Parameter values were derived from estimates of dispersal kernels and life history parameters published in the literature from 11 woody invasive species, from shorter-lived European gorse (*Ulex europaeus*) to long-lived *Pinus* spp. (see Coutts et al., 2011; and appendix 2 of that paper for details). However, these values acted as a guide only. The purpose of this study was to reliably induce invasion lags and we chose parameter values that could reliably induce invasion lags, within realistic ranges. To test the robustness of our results to changes in parameter values, we ran the model under factorial combinations of high and low values for each parameter. See Table 1 for parameter values used and the levels for the sensitivity analysis, and Appendix S1 for more details of the sensitivity analysis.

2.1 | Landscape, scales and dispersal

Invasion lags are scale-dependent phenomena. A new population might fill a small patch very quickly, with no invasion lag at the patch scale.

However, if we consider the whole landscape, a population may be trapped in a small patch, so there is an invasion lag at the larger scale. We run the model on a 128×128 cell grid, a scale that is computationally tractable and that allows the two specific mechanisms we built into the model to generate lag phases (changing dispersal and fragmented habitat). The landscape has reflecting boundaries so that the structure of the gaps is preserved, but seeds are not lost over the side of the landscape.

We generate landscapes where clumps of suitable habitat are separated by gaps of unsuitable habitat using a fractal generating algorithm. Following With, K. A. (1997), we used a diamonds and squares midpoint displacement algorithm. This algorithm has a clumping parameter that we set to 0.5, a value that results in continuous patches of suitable habitat separated by wide gaps. It is these gaps that cause the invasion lags. We influence the gap size by reducing the amount of suitable habitat (r). This is consistent with habitat fragmentation caused by land conversion and increases the gap between suitable patches while reducing the size of those patches. Henceforth, we use the term fragmentation to describe landscape structure, to emphasize the role that gaps play in causing the invasion lags. Technically, both amount of suitable habitat and the arrangement of that habitat (clumping) influence fragmentation.

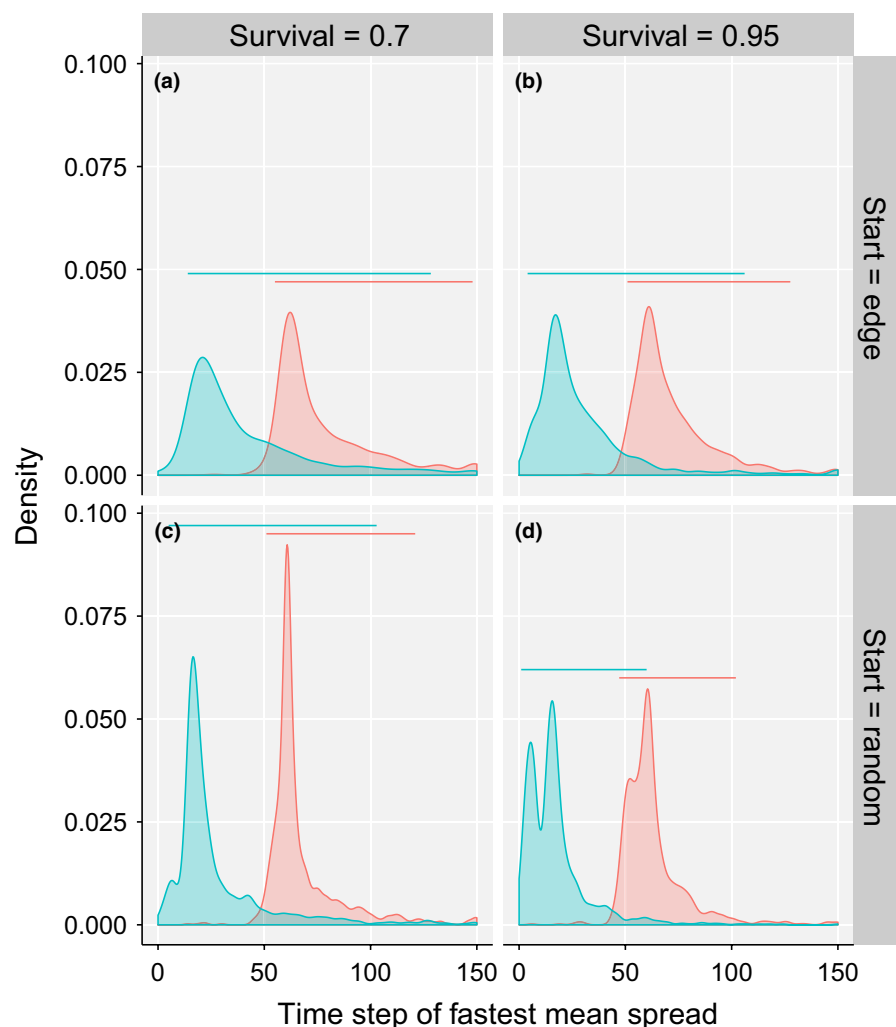


FIGURE 3 Time step of the fastest mean spread rate in the absence of a change in dispersal kernel (blue) and when the dispersal kernel changed from restricted to less restricted after 50 time steps (red), under different starting conditions (rows) and survival probabilities (columns). Age of first seed production is 3 time steps, and new established plants per adult per time step (effective fecundity) is 32% and 25% of cells are suitable. Bars show the 95% quartiles for each distribution. Frequency distributions exclude invasion trajectories that occupied <10% of the suitable cells after 150 time steps. [Colour figure can be viewed at wileyonlinelibrary.com]

Gaps are only relevant to the scale of dispersal, because they can only cause invasion lags if it is unlikely that propagules will cross those gaps. Dispersal is controlled by a 2Dt dispersal kernel (Clark et al., 1998). The 2Dt kernel is flexible and has well-known kernels as special cases, see and Appendix S1 for more details. The kernel has two parameters: the mean dispersal distance (μ) and a shape parameter that controls the frequency of long-distance dispersal (c).

We use three different dispersal kernel parameterizations in this analysis (Figure 1). The restricted dispersal kernel (amber, Figure 1) has a short mean dispersal distance ($\mu = 1$) and shape parameter ($c = 2$) that results in a thin tail. Under this parameterization, the kernel is Gaussian, leading to diffusion dispersal. The restricted kernel is used to induce invasion lags. The less restricted kernel ($\mu = 3$, $c = 0.5$; blue, Figure 1) is a fat-tailed kernel, where long-distance dispersal is more likely than the under the restricted kernel (Figure 1d). We use the less restricted kernel to break invasion lags as its fat tail makes it more likely that propagules will cross gaps. However, gaps are still relevant as crossing them is a probabilistic event that may take some time. To demonstrate the interaction between fragmentation, landscape size and dispersal scale, we run a high dispersal scenario ($\mu = 10$, $c = 0.5$; black, Figure 1) as part of the sensitivity analysis. At the scale of our landscape, the high dispersal kernel allows propagules to cross gaps easily.

2.2 | Simulation experiments

We generate lag phases through a change in the dispersal kernel. For the first 50 time steps, the invaders move according to either the restricted dispersal kernel (amber, Figure 1) or a less restricted kernel (blue, Figure 1). After 50 time steps, each dispersal kernel is either kept the same or changed to the less restrictive kernel, for a further 100 time steps or until 95% of the suitable cells are occupied.

To test the effect of the starting location on the role fragmentation plays in making invasion lags less predictable, we start the invasion in either a random cell or at one edge of the landscape (centre-most cell on the southern edge). When the invasion starts at the edge of the landscape, initially it must cross gaps sequentially, filling each patch before crossing to the next patch. In contrast, when the invasion starts in a random cell, it is likely to begin in the interior of the landscape, and so initially the population can disperse propagules across gaps in all directions simultaneously. This will provide more chances to cross all the gaps in a landscape over the 150-time step run.

The break-in invasion lag is the moment the invader has the greatest potential to spread; the restriction that was holding it back is released, and a significant amount of the landscape is still available for colonization. We assume this break corresponds with the invaders' fastest sustained spread rate. We identify the time step with the fastest spread rate by calculating the moving average spread rate (over a 21-time step window) at each time step, to smooth out small variations (see Appendix S1). Visual inspection showed that this measure corresponded well with the timing of large, sustained changes in population spread (i.e., breaking invasion lag).

3 | RESULTS

On average, when the dispersal kernel changed, invasion trajectories were consistent with release from a lag phase caused by a change in the dispersal kernel (sharp rise in black line, Figure 2c,d). However, among individual runs, invasion trajectories were unpredictable in fragmented landscapes. While many runs under the change in dispersal showed clear population increases immediately following the dispersal kernel change, the growth rate of many other runs increased much later (Figure 2c–f). Across all runs, this variability obscured the link between the change in the dispersal kernel and the break-in the invasion lag. Compare the overlap of distributions when the dispersal kernel changes (red) and when it does not (blue) in Figure 3.

In addition, in several control runs where only the less restricted dispersal kernel was used (i.e., there was no change in model parameters), the population began to expand around the 50-time step point, corresponding with the change in dispersal kernel in non-control runs (Figures 2e,f and 3). This was especially true if the invasion started at

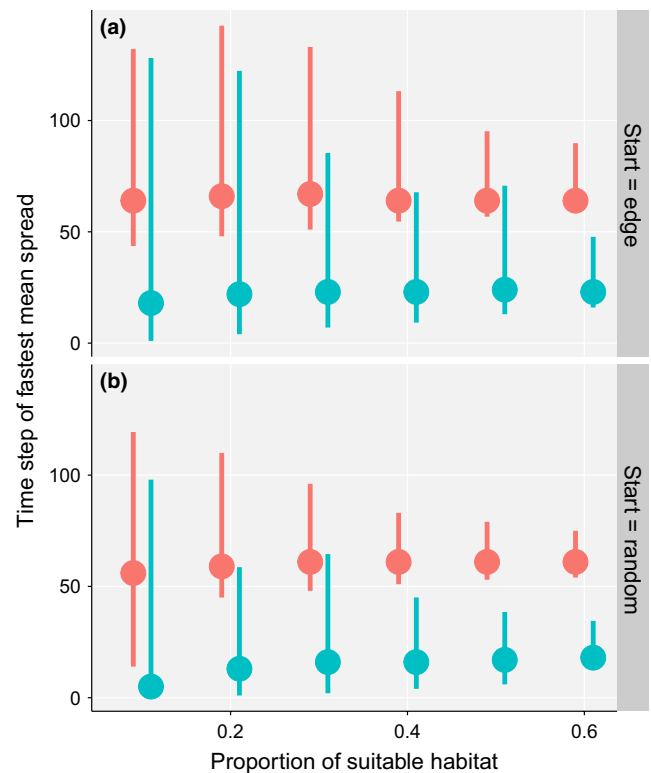


FIGURE 4 Median time step of fastest mean spread rate over different levels of fragmentation (dots) in the absence of a change in dispersal kernel (blue) and when the dispersal kernel changed from restricted to less restricted after 50 time steps (red). Bars show the 95% quartiles for each distribution. The population was started from an initial suitable cell that was randomly chosen, or the centre-most cell on an edge of the landscape. Age of first seed production is 3 time steps, and new established plants per adult per time step (effective fecundity) is 32 and survival probability is .95. Excludes invasion trajectories that occupied <10% of the suitable cells after 150 time steps. [Colour figure can be viewed at wileyonlinelibrary.com]

the edge of the landscape (Figures 2b,d,f and 3), exaggerating the ability of gaps to cause invasion lags, although there was still wide variation in invasion trajectories when the invasion started in a random cell (Figure 2a,c,e).

Even in this highly simplified model system, there were strong interactions between demographic parameters and the starting location of the invasion in fragmented landscapes. When the invasion started at the edge of the landscape, reducing survival probability from .95 to .7 did not greatly change the distributions of time step of fastest average spread (Figure 3a,b), although there was greater uncertainty when survival was lower (distributions have fatter tails in Figure 3a vs. b). When the distribution started in a random cell and survival probability was .95, the tails of the distributions of time step of fastest average spread were thinner and there was less overlap between the distributions when the kernel changed (red) and when it did not (blue), Figure 3d. When survival probability was .7, the distributions had long, fatter tails (compare distributions in Figure 3c), which resulted in a large overlap between the distribution when the kernel changed (red) and when it did not (blue). However, these distributions were also very peaked, so that the bulk of the distribution lies over a narrow range of time steps (Figure 3c).

The amount of fragmentation had a large effect on variability of the length of the invasion lag, both when the dispersal kernel changed and when it did not (red and blue, Figure 4). As expected, the effect of fragmentation was greater when the invasion started on the edge of the landscape, rather than in a random cell. When the invasion started

at the edge of the landscape, the distribution of invasion lag times under the control runs (i.e., kernel did not change) overlapped with the median of the distribution of invasion lag times when the kernel changed, even when up to 50% of the landscape was suitable habitat (Figure 4a). When the invasion started in a random cell, this degree of overlap only occurred when up to 30% of the landscape was suitable (Figure 4b).

When the initial invasion was forced to start in the centre-most suitable cell, these results were qualitatively unchanged from those when the invasion started in a random cell (see Appendix S2).

The sensitivity analysis showed these general patterns were robust to changes in parameter values; however, the details of population trajectories did change. The sensitivity analysis revealed three general patterns (Figure 5, Appendix S1). In the first pattern, there was lower variation between replicate invasion trajectories and a clear invasion lag in the dispersal limitation release scenario. This pattern occurred when the landscape was better connected (e.g., $r = .5$; Figure 5a) or when the dispersal kernel allowed high levels of long-distance dispersal (e.g., $\mu_2 = 10$; Figure 5c). In the second pattern (Figure 5b), there was less dispersal limitation (many of the grey lines increased before the kernel changes) due to an early age of maturity ($m = 1$). However, due to the effect of habitat fragmentation ($r = .25$), there was still considerable variation in invasion trajectories. The third general pattern we observed was characterized by a strong effect of dispersal limitation and high variability between replicate invasion trajectories (Figure 5d). This pattern occurred when age of

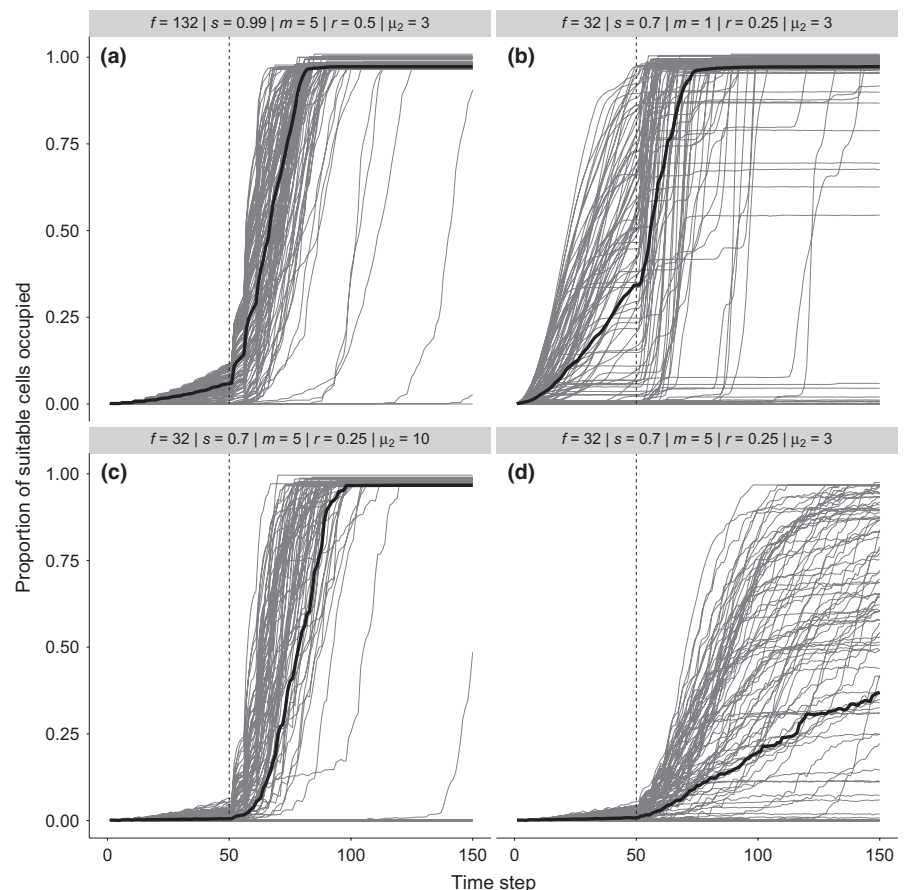


FIGURE 5 Population trajectories for selected parameter combinations from the sensitivity analysis (a–d). Parameters varied are effective fecundity (f), survival probability (s), age of first seed production (m), proportion of suitable habitat (r) and mean dispersal distance of the second dispersal kernel (μ_2). All runs start at the edge of the landscape under the restricted dispersal kernel. At 50 time steps, the dispersal kernel changes to either the less restricted kernel ($\mu_2 = 3$, $c = 0.5$; panels a, b, d) or the high dispersal kernel ($\mu_2 = 10$, $c = 0.5$; panel c). The full set of plots are available in Appendix S1

maturity was later (e.g., $m = 5$), and suitable habitat was fragmented (i.e., $r \leq .5$).

4 | DISCUSSION

Although we could detect a signal of the release from dispersal limitation when results were averaged across many runs, the variability among runs was so great that we could not reliably discern where the dispersal kernel changed. Further, in several control runs, we observed a lag release that was coincident with the dispersal change in experimental runs. These lag breaks occurred with no change in circumstance apart from stochastic spread to an area of larger suitable habitat, a result seen empirically (Bennett et al., 2013).

Population managers tend to be risk-averse (Tulloch et al., 2015). Thus, the fact that we could pick up and predict invasion lags “on average” may be less important to managers than the minority of runs with very long lags. What this means in practice will be context dependent, but in general it will complicate early detection and control strategies (Holden et al., 2016), which require predicting which species will become widespread and invasive. Our results suggest a lack of early spread is an unreliable predictor of a species potential to spread.

An alternative is to use models to predict which species will become invasive. This will likely require multiple complementary approaches. Species distribution models can be used to estimate the amount of suitable habitat available to invade and potentially the degree of fragmentation (Guisan et al., 2013). Weed risk assessment protocols, often employed to screen species before introduction (Leung et al., 2012), may also be used to predict which species already present at low levels have the highest potential to become invasive. Our results also suggest that the use of barrier zones (making land use decisions or using management to impose gaps; Epanchin-Neill & Wilen, 2011; Southwell et al., 2016; Bode et al., 2013) is likely to slow invasion, especially if the invasion is coming in from an edge. However, the length of that delay could be very unpredictable.

We may never be able to predict the timing of invasion lags with reasonable accuracy. This is especially true in highly fragmented landscapes, and when the invasion enters that landscape from the edge, such as when starting from a coastal region or the edge of a forest. Developing tools that optimize decisions under this uncertainty may be the best we can do (e.g., Chadès et al., 2011).

Our results also show that inferring mechanisms from a coincidence in timing bring a considerable risk of false attribution. Indeed, even when we only used the less restrictive dispersal kernel, we still observed considerable invasion lags in some runs. This may reflect an inherent characteristic of expanding populations in fragmented landscapes (Crooks, 2005). The alternative to using coincidence of timing is direct manipulative experiments, which are extremely difficult both ethically and practically; or, if it was believed that a lag phase was broken by a common event for multiple species, comparative approaches could build replication of time series among multiple species.

5 | CONCLUSIONS

Even when there is a clear event that might lead to rapid population expansion, for example a new dispersal vector (Amodeo & Zalba, 2013), our results suggest that it will be very difficult to reliably predict the extent or timing of a rapid invasion. In reality, there are many causes of invasion lags, as well as lags between rapid expansion and detection or observed impacts (Crooks, 2005). These detection lags are likely to make prediction of which species are in a lag phase, and how long that lag phase is likely to last, even harder. We have to acknowledge the uncertainty around invasion lags and adopt management strategies that accommodate it. Given the potential for extremely high costs associated with invasive species that do spread rapidly, we reiterate the recommendation of Crooks (2005) for the precautionary principle in managing potentially invasive species, even if they appear to be relatively benign.

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BIOSKETCHES

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

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